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NEWSLETTER

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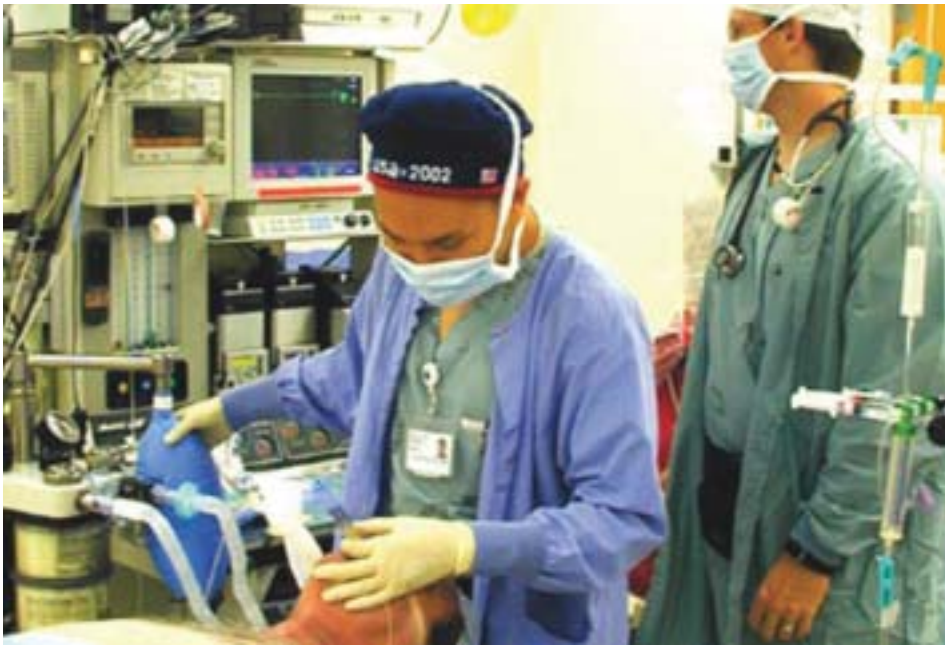
Can We Alter Long-Term Outcome?

The Role of Inflammation and Immunity in the Perioperative Period (Part II)

by Steffen E. Meiler, MD, Terri G. Monk, MD, James B. Mayfield, MD, and C. Alvin Head, MD

Editor's Note: The fall 2003 Issue of this Newsletter contained an article entitled "Can We Alter Long-Term Outcome? The Role of Anesthetic Management and the Inflammatory Response," by Drs. Meiler, Moond, Mayfield, and Head. The following article is the second in this two-part series and provides a glimpse into evidence linking perioperative inflammation and long term morbidity and mortality. It raises some very interesting and provocative questions.

As part of this initiative exploring perioperative inflammation, the APSF is organizing a multi-specialty Expert Panel entitled "Anesthetic Depth, Inflammation, and Surgical Outcomes," to be held in Washington, DC, in September 2004, under the leadership of David Gaba, MD. A world-class group of investigators will meet to consider the current data, identify gaps in understanding, develop research strategies, and discuss the potential impact on perioperative management. Surgical leaders will participate, including Shukri Khuri, MD (VA), Thomas Russell, MD (ACS), and David Hunt (CMS), as well as anesthesiologists and scientists including Charles Serhan, MD, Simon Gelman, MD, Lee Fleisher, MD, Rober Legasse, MD, Marcel Durieux, MD, Steffen Meiler, MD, and Terri Monk, MD. Patient Safety experts will include Robert Stoelting, MD, and Jeffrey Cooper, PhD. This panel will be co-sponsored by the Joint Commission on Accreditation of Healthcare Organizations, represented by Jerold Loeb, PhD, and the National Quality Forum, represented by Kenneth Kizer, MD.



Our short-term interventions may have long-term consequences.

The first article in this two-part series introduced the APSF readership to the concept that medical decisions during the perioperative period may have long-term consequences for patient safety.[1] The potential relationship between anesthesia management and long-term outcome is not intuitively obvious. Anesthesia practice typically functions within a "beat-to-beat" environment where intraoperative complications happen suddenly, anesthetic drugs wear off quickly, and patient outcomes are measured within hours or at most a few days following surgery. Most anesthesia patient safety initiatives have naturally focused on events that occur while anesthesiologists are actively involved with care—a period that has now become extraordinarily safe. This focus may need to shift as the risk of dying during the first postoperative year may be as high as 5% to 14% in certain patient populations. New evidence suggests that this mortality might be influenced by specific anesthetic interventions at the time of surgery.[2-4]

Recent biomedical research has demonstrated the importance of inflammation in the progression of chronic diseases such as atherosclerosis, cancer, and dementia. Anesthesia and surgery are also associated with a dramatically increased inflammatory response and concurrent suppression of cell-mediated immunity.[5,6] Since most long-term deaths after surgery are due to cardiovascular events and cancer, it is reasonable to postulate that perioperative immune responses play some role in these outcomes.

See "Outcome," Page 3

Inside:

A Partnership and Common Vision

The Anesthesia Patient Safety Foundation (APSF) and The Coalition for Critical Care Excellence (CCCE) Initiate Joint Taskforce

Safety initiatives continue to be refined throughout the continuum of perioperative and extended critical care. Patients commonly travel through these areas, and attention to quality care and patient safety should be universal across all providers and throughout the organization. Defining common goals provides opportunity for anesthesia and critical care practitioners to collaborate on best practices and initiate new approaches to optimize patient safety.

Therefore, the Anesthesia Patient Safety Foundation (APSF), founded in 1986, and the Coalition for Critical Care Excellence (CCCE), founded in 1991, have appointed a Joint Taskforce on Safety to explore a shared vision and mission to improve patient safety throughout the perioperative period.

The goals of the Joint Taskforce are to

- Communicate safety priorities, best practices, and related programs conducted by each association.
- Evaluate and implement partnering opportunities for patient safety.
- Provide updates to the leadership of the American Society of Anesthesiologists (ASA) and The Society of Critical Care Medicine (SCCM).

Members of the Joint Taskforce on Safety include APSF appointees, Jeffrey Cooper, PhD; David Gaba, MD; Robert Morell, MD; and Richard Prielipp, MD (co-chair); and CCCE appointees, Yvonne Harter, MBA; Patricia McGaffigan, RN (co-chair); Marcus Schabacker, MD; and Margaret Parker, MD.

For additional information on the Safety Joint Taskforce please contact by email: Richard Prielipp with the APSF at prielipp@wfubmc.edu, or Patricia McGaffigan with Aspect Medical, representing the CCCE, at pmcgaffigan@aspectms.com.

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The APSF Website is now undergoing a redesign—stay tuned for a new, user-friendly, exciting, and informative website!

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Perioperative Inflammation Model Suggested

"Outcome," From Page 1

In this article we discuss some of the research on this topic, and briefly address three broad questions:

1. What is the evidence that perioperative inflammation and immunity are determinants of long-term morbidity and mortality?
2. Do we have evidence that anesthesia care is likely to affect this biology? Is it possible that some anesthetic approaches accelerate disease processes while others have protective effects?
3. Will it be possible, by using these insights, to improve preoperative risk stratification and develop strategies that reduce long-term adverse events?

Inflammation: A Key Element in Disease

"Inflammation is a local, protective response to microbial invasion or injury. It must be fine-tuned and regulated precisely, because deficiencies or excesses of the inflammatory response cause morbidity and shorten lifespan."

— *Nature* 2002;420:853

There is now a large and growing literature on the role of inflammation in the pathogenesis of atherosclerosis and cancer. Atherosclerosis is generally held to be an inflammatory disease of the vascular wall, and it responds to treatment with diverse classes of anti-inflammatory medications, such as aspirin, beta-blockers, HMG-CoA reductase inhibitors (statins), ACE-inhibitors, and activators of PPAR-α (fibrates).[7,8] Statins, for example, have been shown in large trials to reduce mortality, but this benefit cannot be completely explained by their effects on cholesterol. It is likely that some of the benefit comes from their significant vascular anti-inflammatory effects.[9,10] Another body of research suggests a link between inflammation and cancer. Many cancers not only arise from areas of chronic infection and inflammation (e.g., hepatitis/hepatocellular carcinoma or inflammatory bowel disease/colon cancer), but they often require a pro-inflammatory milieu to support their growth and metastatic spread.[11] The fact that chronic use of aspirin and NSAIDs is associated with a reduced incidence of certain malignancies further substantiates the role of inflammation in the biology of cancers.[12]

What about anesthesia and surgery? It is now well recognized that surgery frequently produces profound changes in both the innate and acquired immune systems.[5,6] The duration and the extent of a surgical procedure as well as preoperative anxiety and postoperative pain can all influence this response. In the perioperative period, there is a heightened inflammatory state, orchestrated by the release of pro-inflammatory cytokines, reduced levels of anti-inflammatory cytokines, and increased production of arachidonic acid metabolites, superoxide radicals, and other mediators. Interestingly, individual genetic profiles, assessed by gene polymorphism, may also predispose certain patients to an enhanced inflammation risk.[13] To put this concept into perspective, we propose a hypothetical model to explain how the perioperative immune response might impact long-term outcomes (Figure 1). This model is supported by the following rationale and studies that document the postoperative progression of disease known to be influenced by inflammatory processes:

1. The typical pro-inflammatory mediators released in surgical patients overlap significantly with those involved in atherosclerosis and cancer.[5-9]
2. The beneficial effects of beta-blockers on postoperative cardiac risk [14] were initially attributed to altered hemodynamics; however, more recent studies demonstrated their significant anti-inflammatory properties.[15,16]
3. Surgical patients demonstrate reduced lymphocyte counts and function, which could result in impaired tumor cell surveillance and elimination. Several studies have reported on the association of surgery and an increased incidence of metastatic tumor spread.[17-19]

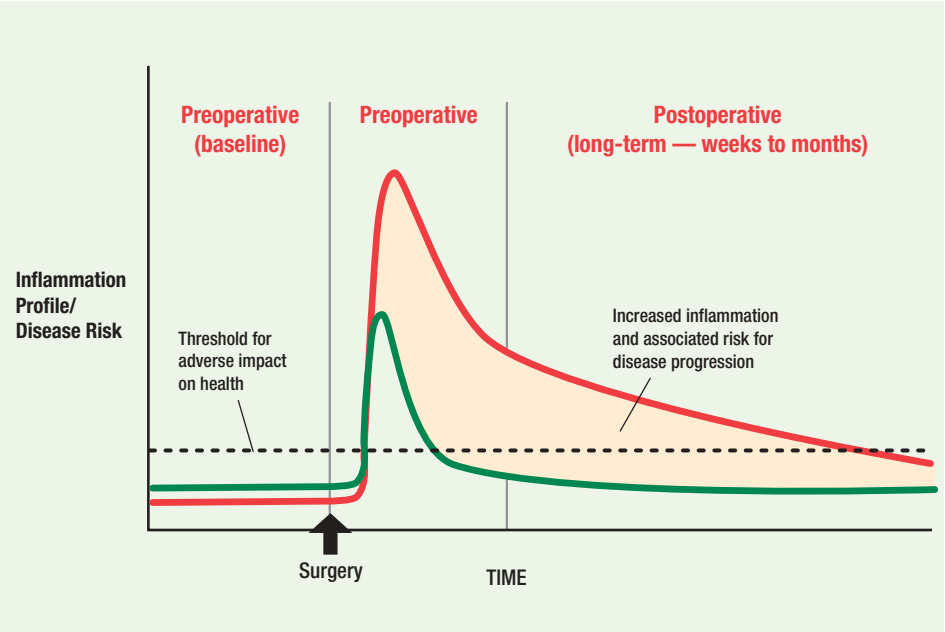

Figure 1. *Perioperative Inflammation and Long-Term Risk: A Hypothetical Model.*

The green curve depicts the perioperative inflammatory response of an otherwise healthy individual, characterized by a transient elevation in various inflammatory mediators and then prompt return to a baseline state. In a high-risk patient (red curve), the amplitude and duration of the inflammatory response are prolonged. This sustained period of inflammation may contribute to new or accelerated disease risk.

Possible Changes in Perioperative Management: Considering the Immune System

At this point, the link between patient outcome and immune response is circumstantial, but credible. Enough is known to hypothesize a variety of clinical interventions that might improve or protect immune homeostasis (see Figure 2).

- The neuroendocrine response to stress is an important modifier of immune function, and anxiety, fear, and pain have been shown to be associated with adverse outcomes.[20] There is a wealth of data on modification of stress hormone responses by anesthetic drugs, but very few studies relating hormone levels to long-term outcome. Stress hormones like norepinephrine can trigger a pronounced and immediate activation of pro-inflammatory cells and cytokines.[21,22] The triggers for stress responses are both psychological and physical, so it seems reasonable that postoperative immune function could be improved by increased attention to perioperative anxiolysis and analgesia.
- Once adrenergic stress hormones have been released, their pro-inflammatory signals can be effectively intercepted with the use of beta- or alpha-blockers.[22] Despite well-documented

See "Outcome," Next Page

Research Needed to Assess Long-Term Outcome

"Outcome," From Preceding Page

benefits for short- and long-term outcome, the perioperative use of beta-blockers remains disconcertingly low (the topic was previously discussed in this newsletter[23,24]). Given the success of beta-blocker treatment, similar outcome studies using other anti-inflammatory drugs (statins, COX-2 inhibitors, ACE-inhibitors, local anesthetics) deserve high priority.

- Perioperative temperature management is now known to play an important role in postoperative inflammatory processes. Maintenance of intraoperative normothermia improves wound healing and reduces wound infection,[25] while mild hypothermia is known to be associated with lymphocyte suppression and increased stress hormone levels.[26,27]
- A small number of studies indicate that certain anesthetic agents exert direct toxic effects and may contribute to long-term risk. In some clinical studies, volatile anesthetics have been associated with a greater systemic inflammatory response compared to total intravenous anesthetics with adjuvant narcotic therapy.[28,29] In vitro studies have shown that volatile anesthetics have substantial immunosuppressive effects by inducing programmed cell death in lymphocytes, diminishing lymphocyte function, and altering the distribution of lymphocyte cell subsets.[30-33] In a study of metastatic melanoma in the mouse, volatile anesthetics alone (without surgery) caused a 2- to 3-fold increase in tumor burden.[19] It is too early to link the immune-modulating effects of volatile anesthetics to an increased risk for tumor growth and spread in humans, but further work in this area is clearly needed.
- The studies of Weldon and Lennmarken show that deeper levels of anesthetic effect and, presumably, larger average doses of volatile anesthetics are associated with increased risk of mortality.[2,3] This suggests a possible patient benefit from reducing the overall exposure to inhaled anesthetic agents. The anesthetic concentration needed to prevent consciousness (MAC-awake) is much less than that needed to prevent movement (MAC) or autonomic responses (MAC-BAR).[34] Titrating volatile agents to suppress movement or to blood pressure and heart rate endpoints, not only results in the administration of higher doses, but may also be associated with underutilization of adjuvant therapy, such as beta-blockers and opioids. When anesthetic agents are titrated to hypnotic endpoints using EEG-based monitors, there is a significant reduction in anesthetic dosing.[35-37]

Regardless of the interventions we ultimately choose to investigate, it will be important to "measure" inflammation by analysis of appropriate serum markers. Measuring inflammation at preoperative baseline may significantly improve the accuracy with which we stratify patients for operative risk. The markers of inflammation, C-reactive protein and myeloperoxidase, have a high predictive utility in determining the clinical course and acute event rate of coronary artery disease.[38,39] Preoperative measurements of these markers may provide a "window" on the inflammatory state or vulnerability of coronary lesions and improve outcome predictions. It is also possible that a postoperative profile of these bio-markers may identify patients who would benefit from more sustained anti-inflammatory treatment strategies. Similarly, these postoperative profiles may provide a means for optimizing anesthetic management.

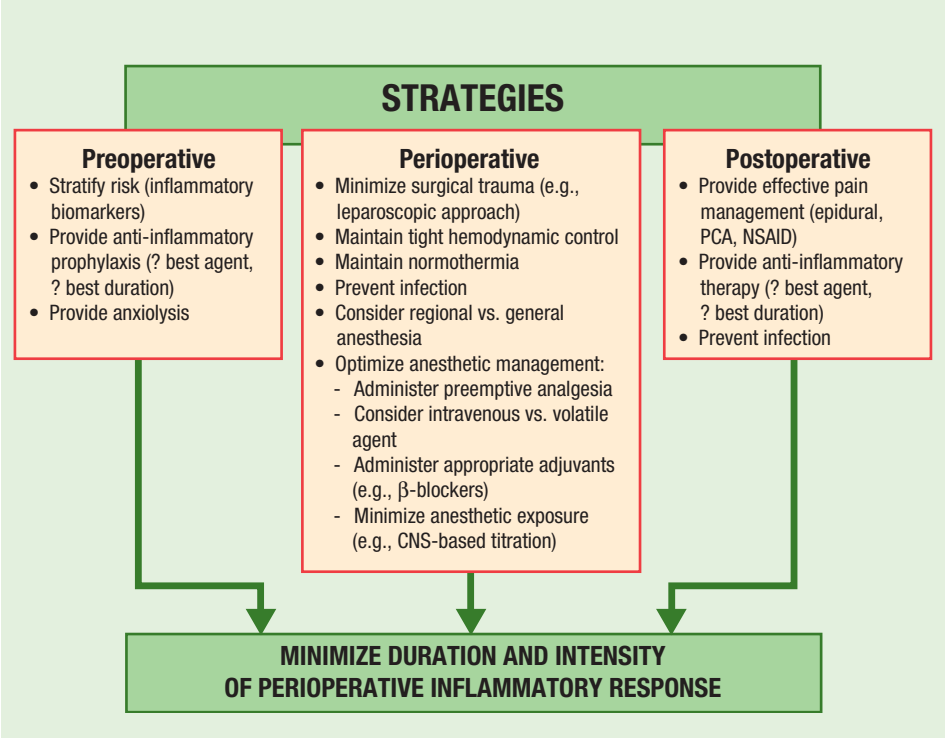

Figure 2. Conceptual framework of strategies that may minimize the perioperative inflammatory response and long-term adverse outcomes.

In summary, the available evidence strongly suggests that immune system dysfunction in the perioperative period, with its combined pro-inflammatory and immuno-suppressive effects, can influence long-term disease progression, morbidity, and mortality. A substantial amount of research — much of it, we hope, by members of the APSF — is needed to establish whether changes in anesthetic practice can meaningfully reduce this risk. Should anesthesiologists really be looking at events 6 months or a year following surgery? The demographics are certainly compelling, since even a small improvement in 1-year outcome could mean thousands of lives saved each year and a significant reduction in economic burden. Although some of the observations shared in this article are still at the conceptual stage, we are confident that research on perioperative inflammation and immunity will yield significant improvements in long-term patient safety.

Dr. Meiler is an Associate Professor and Vice Chair for Research in the Department of Anesthesiology and Perioperative Medicine, and Director of the Program for Molecular Perioperative Medicine & Genomics at the Medical College of Georgia. Dr. Monk is a Professor in the Department of Anesthesiology at the University of Florida College of Medicine. Dr. Mayfield is an Associate Professor and Vice Chair of Clinical Anesthesia in the Department of Anesthesiology and Perioperative Medicine at the Medical College of Georgia. Dr. Head is Professor and Chair of the Department of Anesthesiology and Perioperative Medicine at the Medical College of Georgia.

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See "Outcome," Page 7

Complications Associated With Regional Anesthesia

Review of ASA Closed Claims

by Lorri A. Lee, MD

Many anesthesiologists perceive regional anesthesia to be a safer alternative to general anesthesia because it has been associated with reduced postoperative mortality caused by thromboembolic disease and myocardial infarction, improved postoperative analgesia, decreased incidence of early postoperative cognitive dysfunction, and shorter recovery times compared to general anesthesia.[1,2] However, significant morbidity may directly result from regional anesthesia. The incidence of cardiac arrest associated with spinal blockade has been reported to be as much as 0.06%, and frequently results in death or brain damage.[3-5] Other high severity complications associated with regional anesthesia include epidural hematoma, cauda equina syndrome, and unintentional intravenous injections of local anesthetic. Less severe injuries such as postdural puncture headache, inadequate analgesia, transient paresthesias, and back pain are more frequent sequelae of regional anesthesia. To determine the differences in liability claims associated with regional anesthesia compared to claims associated with other types of surgical anesthesia (general anesthesia and monitored anesthesia care), we utilized the database of the American Society of Anesthesiologists (ASA) Closed Claims Project. Because the database does not contain denominator data on all anesthetics performed, this analysis does not provide information comparing the incidence of injuries between regional and general anesthetics, but offers a perspective on the liability associated with each group. Claims between 1980 to 1999 that were associated with regional anesthesia and other surgical anesthesia were included in this analysis from a database of 5,802. Chronic pain blocks, postoperative pain blocks placed outside the operating room, eye blocks performed by ophthalmologists, and obstetric claims that involved no maternal injury were excluded. A more detailed analysis of regional anesthesia claims including eye blocks and peripheral nerve blocks, as well as a subset comparison of obstetric and non-obstetric neuraxial claims is reported elsewhere.[5]

Obstetrics was associated with approximately one-third (36%) of the regional anesthesia claims (n = 1005) and significantly influenced the demographics of this group, resulting in significantly more ASA 1-2, young, female patients compared to the other surgical anesthesia group (n = 3551). Epidural anesthesia was utilized most commonly in regional anesthesia claims (42%), followed by spinal blockade (34%), axillary blocks (6%), eye blocks (4%), interscalene blocks (2%), and intravenous regional anesthesia (IVRA).

The regional anesthesia group contained a significantly higher proportion of claims associated with temporary injury and permanent nerve injury compared to the other surgical anesthesia group, whereas death or permanent brain damage was present in a significantly higher proportion of other surgical anesthesia claims (Figure 1).

The primary damaging events (mechanism by which the injury occurs) were block-related in 48% of regional anesthesia claims (Table 1). Complication of block technique was the most common damaging event in these claims.

Regional anesthesia claims with death or brain damage were associated with block-related primary damaging events in 49% of cases. Of these events, neuraxial cardiac arrest (i.e., the sudden onset of severe bradycardia or cardiac arrest during neuraxial block with relatively stable hemodynamics preceding the event) was the most common (37%), followed by unintentional intravenous injection (6%), and other block/anesthesia-related events (6%). In death or brain damage claims associated with regional techniques, the breakdown was as follows: 51% spinal blockade, 41% epidural, 2% interscalene block, 1% axillary block, and 5% miscellaneous blocks.

Eighty-four percent of the neuraxial cardiac arrest claims from the 1980s and 1990s (n = 81) were associated with either intentional (70%) or inadvertent intrathecal blockade (14%). The remainder of these claims was associated with lumbar epidural (12%), caudal epidural (2%), and thoracic epidural (2%) anesthesia. Outcome for neuraxial cardiac arrest resulted in death or brain damage in 91% of cases, and was similar between decades. The use of pulse oximetry and capnography in the 1990s claims (n = 31) did not prevent the occurrence of neuraxial cardiac arrest or improve the outcome of death or brain damage compared to the 1980s claims when these monitors were not widely available (Table 2). It is unclear if prevention or resuscitation of neuraxial cardiac arrest has improved over the last decade because the ASA Closed Claims Database lacks denominator data and is biased toward cases with poorer outcomes. Inadvertent intrathecal blocks and occurrence of cases outside the operating room may account for some of the delays in recognition and resuscitation of neuraxial cardiac arrest. The rapid onset of bradycardia/asystole and the intense loss of sympathetic tone caused by spinal blockade (which reduces circulating blood volume) may explain why some cases of neuraxial cardiac arrest may be refractory to prompt treatment.

A greater proportion of regional anesthesia claims were associated with permanent nerve injury compared to the other surgical anesthesia group (Figure 1). Lumbosacral nerve root, paraplegia, and median nerve damage were significantly more common in the regional anesthesia group compared to the other surgical anesthesia group. Brachial plexus, ulnar nerve, and femoral/sciatic nerve injuries occurred in a greater proportion of other surgical anesthesia claims compared to regional anesthesia claims. Eighty-four claims of injuries to the neuraxis were identified in the regional anesthesia group, and 36 cases (44%) were associated with hematoma. Outcomes for neuraxial injuries, including hematoma, anterior spinal artery syndrome, spinal cord infarct, and other/unknown causes, were poor and resulted in permanent neurologic damage in the majority of cases. Neuraxial injury caused by herniated discs and infections such

See "Complications," Next Page

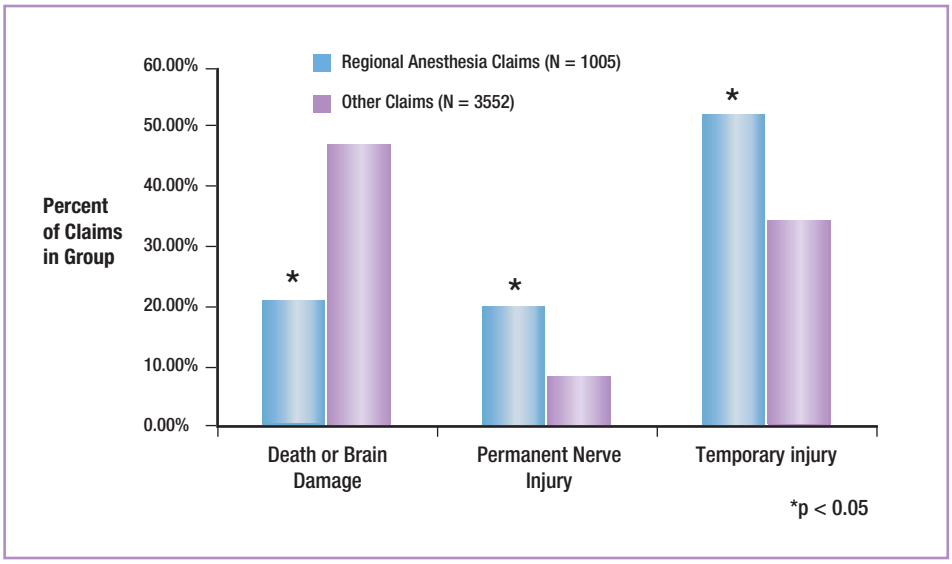

Figure 1: *Outcomes in Regional Anesthesia Vs. Other Surgical Anesthesia Claims 1980-1999.*

Regional Accounts for 20% of Claims

"Complications," From Preceding Page

as meningitis and abscess demonstrated full recovery in the majority of cases. Vascular surgery was the most common operation associated with hematoma (56%), and coagulopathy was associated with 72% (n = 26) of all hematoma claims. Needle trauma above L1 occurred in 17% of hematoma claims with neuraxial injury. The most common presenting symptom was increased motor block out of proportion to the local anesthetic used (83%), followed by increased sensory block (53%), and back pain (25%). A median delay of 1 day from symptom onset to diagnosis was thought to contribute to the poor neurologic recovery in the majority of hematoma claims. Heightened vigilance for symptoms and signs of epidural hematoma (e.g., increased motor block) and prompt diagnostic work-up and treatment may improve neurologic outcome.

Claims associated with regional anesthesia had a significantly lower percentage of claims with payment to the plaintiff compared to the other surgical anesthesia group, a lower median payment, a higher percentage of claims judged to have met appropriate care standards, and a lower percentage of payment in cases with appropriate standard of care. These differences may reflect the greater proportion of regional anesthesia claims with temporary injury compared to other surgical anesthesia claims.

In summary, regional anesthesia accounts for approximately one-fifth of professional liability claims. Most of the injuries from regional anesthesia claims are temporary, and approximately one-half of the temporary claims are associated with obstetrics. High severity injuries continue to result from neuraxial cardiac arrest and neuraxial hematomas associated with coagulopathy. Heightened vigilance and prompt diagnosis and treatment may improve outcome in these cases.

Dr. Lee is an Assistant Professor in the Department of Anesthesiology at the University of Washington, Seattle, WA.

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Table 1. Block-Related Primary Damaging Events in Regional Anesthesia Claims

	n (%)
Block-related	**482 (48%)**
Block technique	249 (25%)
Neuraxial cardiac arrest	81 (8%)
Inadequate anesthesia/analgesia	48 (5%)
High spinal/epidural	41 (4%)
Epidural/spinal catheter	35 (3%)
Unintentional IV injection	28 (3%)
Non-Block-related	**523 (52%)**

Table 2. Associated Factors: 1990s Neuraxial Cardiac Arrest Claims (n=31)

Pulse oximetry	**74%**
Capnography	**35%**
Sedation	**52%**
Out of operating room	**35%**
Recognition delay	**26%**
Resuscitation delay	**61%**

Burton A. Dole, former Vice President of the Anesthesia Patient Safety Foundation.

Burton Dole Becomes Largest Individual APSF Donor

Burton A. Dole, former Vice President of the Anesthesia Patient Safety Foundation, has made yet another important contribution to the APSF. Mr. Dole has designated the APSF to be the recipient of a $200,000 contribution upon the settlement of his estate. This tremendously generous example of planned giving is appreciated by the APSF Executive Committee. Mr. Dole hopes that his gift will serve as an example for others to follow when contemplating estate planning.

Burt was a founding member of the APSF. The Puritan-Bennett company, of which Burt was the CEO at the time, gave the APSF $300,000 in startup funds. The Parker B. Francis Foundation, the charitable arm of the founder of Puritan-Bennett, also gave $300,000 as a founding grant. Burt has served as APSF Treasurer, as well as Vice President, and received the first APSF award for service to this organization in 1997. He retired from the APSF Executive Committee in 2002, but remains on the Board of Directors. His career has been a shining example of a life devoted to helping others and insuring patient safety. *Thank you, Mr. Dole.*

Erratum

On page 58 of the Winter 2003-04 issue of this *Newsletter*, credit to Dr. C.F. Ward, San Diego, CA was omitted from his letter entitled "Power Interruption: Still A Major Safety Disruption." We apologize for this oversight, and appreciate Dr. Ward's thoughtful contribution to the *APSF Newsletter*.

Outcome References Support Need for More Research

"Outcome," From Page 4

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Misplaced Valve Poses Potential Hazard

Michael Olympio, MD, Chair of the APSF Committee on Technology and Co-Founder of the SIRS Initiative.

The APSF is pleased to announce an important new technology initiative which will appear as a regular column in the *APSF Newsletter*. This feature is entitled "Dear SIRS" and refers to the Safety Information Response System. The purpose of this column is to expeditiously communicate technology-related safety concerns raised by our readers, with input and responses from manufacturers and industry representatives. This process was developed by Drs. Michael Olympio, Chair of the Committee on Technology, and Robert Morell, Editor of this newsletter. Dr. Olympio is overseeing the column and coordinating the readers' inquiries and the responses from industry. "Dear SIRS" makes its debut in this issue with an important safety concern raised by James M. Berry, MD, and Steve Blanks, CRNA, from Vanderbilt University Medical Center.

Dear *SIRS*:

We have discovered what we believe to be an error in the assembly of 9 Datex-Ohmeda Aestiva anesthesia machines recently delivered and installed which, if not detected, has the potential to cause injury to anesthetized patients.

The problem is in the AGSS (active gas scavenging system) option which produces, when the evacuation hose becomes occluded, sustained airway pressures (PEEP) of up to 40 cm. This condition is exacerbated by high fresh gas flows and when mechanical ventilation is in use.

The AGSS is designed to have an opening in the bottom of a plastic receiver, providing relief of both positive and negative excess pressures. In what appears to be an assembly error, a negative pressure relief valve (similar to a circle system one-way valve) was installed in this opening (see photos 1 and 2). This provided relief of excess negative pressure (too much evacuation suction), but no positive pressure relief. This valve is used appropriately in the passive system, but the passive system also has a positive pressure relief in the upper portion of the receiver unit. Our best explanation is that the receiver assembly is composed of a top and a bottom section. We were (possibly) delivered units with the top receiver half from an active system and the bottom half from a passive system.

SAFETY
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No injury has occurred in our institution as a result of this incident, but this discovery followed a number of our anesthesia staff reporting unusual ventilator behavior which we subsequently traced to low evacuation flows. The most dramatic incident was when, after a bed rolled over a gas scavenging hose, the Aestiva ventilator immediately stopped cycling in mid-exhalation. The evacuation hose (color coded purple) is equipped with a needle valve which is adjusted to provide moderate flows (as shown by a small ball indicator on the side of the absorber assembly). The presence of the mis-assembled scavenging receiver may be detected either by a temporary ventilator failure when the evacuation hose is manually occluded (hand crimped) or by gross fluctuations of the ball indicator during normal ventilator cycling.

The Datex-Ohmeda company has been prompt and thorough in their response to this incident. They are in the process of identifying AGSS equipped units that may have been assembled incorrectly and are currently pursuing action to remedy this situation.

James M. Berry, MD
Professor

Steve Blanks, CRNA
Chief CRNA
Nashville, TN

***Photo 1**. Bottom view of the active scavenging reservoir without valve (correct configuration).*

***Photo 2.** Bottom view of the active scavenging reservoir with valve in place (incorrect configuration).*

Manufacturer Provides Prompt Response

(In Reply)

Dear *SIRS*:

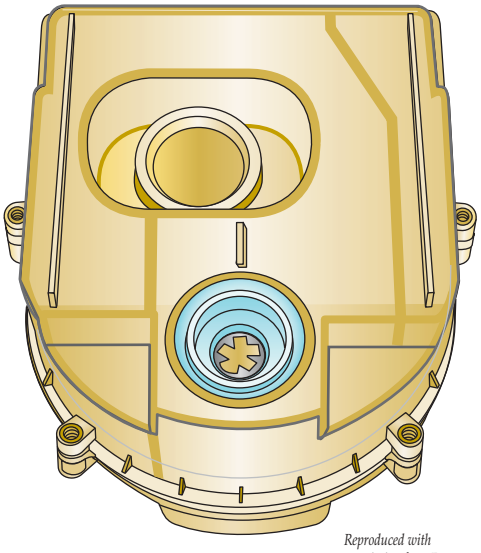
Datex-Ohmeda would like to thank the *APSF Newsletter* for the opportunity to respond to the letter by Berry and Blanks.

The authors have correctly identified the root cause of the rising airway pressure encountered in some of their Aestiva anesthesia machines. There are two options for gas scavenging in the Aestiva. One is passive and the other, the option used by the authors, is an active system that uses vacuum to remove the scavenged gases from the patient circuit and from the ventilator drive system.

The AGSS units that were assembled incorrectly had a valve normally used for the passive systems installed in the base of the AGSS unit. The presence of this valve prevented excessive gases within the AGSS from escaping when the vacuum outflow was occluded, as described by the authors.

Datex-Ohmeda has identified the cause of the incorrect assembly and has instituted changes in the assembly process to avoid a repeat of this error. In addition, there is now an additional test of the AGSS to verify that all units have the correct valves in place. Datex-Ohmeda has also identified the entire population of AGSS units that may have been assembled incorrectly, has identified the location of the entire suspect population, and has begun an active Field Action to check the AGSS units, verify their proper manufacture and assure that any occlusion of the AGSS exhaust hoses will not produce a rise in the system airway pressure in the future.

Bottom View of Active Scavenging Reservoir



Reproduced with permission from Datex-Ohmeda, Madison, WI

Drawing of the bottom view of the active scavenging reservoir without the valve (correct configuration).

Michael Mitton
Director of Clinical Affairs
Datex-Ohmeda, now a part of GE Medical Systems

APSF Spotlight

George A. Schapiro, Executive Vice President and member of the Executive Committee of the Board of Directors of APSF.

APSF Executive Vice President
George A. Shapiro

George A. Schapiro, Executive Vice President of APSF since October 2003, also serves on the Executive Committee of the Board of Directors. He first served on the Board from 1985 to 1991. He has been active in strengthening the APSF Corporate Advisory Council since its inception and served in the late 1980s as Chairman of the APSF Development Committee. He is a management consultant to or a member of the Board of Directors of a number of technology companies, most of which concentrate in medical device businesses. From 1992 to 2001 he served as Interim CEO for six medical device, industrial technology, and biotech companies. From 1976 to 1991, he was President and CEO of Andros Incorporated, supplier of gas analyzers to the patient monitoring industry. From 1969 to 1976, he was with the Hewlett-Packard Company where from 1974 to 1976, he was responsible for the Patient Monitoring product line (now part of Philips Medical Systems) on a world-wide basis.

2005 APSF Grant Applications Due June 14, 2004

See the APSF Website at apsf.org for details!

Letter to the Editor:

Spinal Anesthesia in the 21st Century?

To the Editor:

I read with interest the letter by Dr. J. Antonio Aldrete in your Winter *APSF Newsletter*.[1] He summarized some important aspects of potential problems with drugs and spinal anesthesia. Drugs can certainly cause damage, but one should not forget that unwanted material can also be introduced inadvertently into the subarachnoid space, e.g., a hair follicle.[2]

John Brock-Utne MD, PhD
Stanford, CA

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APSF Scientific Grant Program: Improving Patient Safety Through Research Funding

by Karen L. Posner, PhD

As part of its objective to enhance anesthesia patient safety, the APSF has been providing funding for patient safety research since 1987. The grant program started at a time when funding for patient safety research was virtually non-existent. The objective of the grant program was (and still is) to stimulate studies leading to prevention of mortality and morbidity resulting from anesthesia mishaps. Priority is given to studies that address problems affecting relatively healthy patients or studies that are broadly applicable and promise improved methods of patient safety with a defined and direct path to implementation into clinical care. Priority is also given to studies investigating innovative methods of education and training to improve patient safety.

The APSF Scientific Evaluation Committee conducted a comprehensive evaluation of the APSF Grant Program. This evaluation included a survey of prior grant applicants plus a literature search and grant applicant survey to create a comprehensive list of publications stimulated by the APSF Grant Program.

Between 1987 and 2001, a total of 272 researchers applied for funding for 347 patient safety research projects through the APSF Grant Program. An average of 23 projects were reviewed by the APSF Scientific Evaluation Committee annually. APSF funding was restricted to 2-3 grants per year. Over the first 15 years of the APSF Grant Program, 48 projects received funding from the APSF, for a funding rate of approximately 1 in 7 applications. An anonymous survey was sent to all prior applicants who could be located (237). Responses were received from 95 prior applicants (40%), including 33 who received APSF funding (69% response rate), and 84 who did not receive funding (33% response rate).

Evaluation Results

Stimulating Patient Safety Research

The APSF is meeting its goal of stimulating anesthesia patient safety research. The APSF funded 48 patient safety projects. Nearly all funded studies were completed, with 88% resulting in publications. Two-thirds of grant recipients who responded to the survey indicated that they went on to conduct additional research along the same line of inquiry as their APSF grant. Half received additional funding, totaling over $11 million. There is also evidence that the APSF Grant Program may have stimulated research beyond the projects directly funded by APSF. Nearly half of survey respondents who did not receive APSF funding for their projects went on to complete their projects, with 31% indicating that the APSF application process helped them get started. Most of those applicants still actively conduct anesthesia research.

> "The APSF funding opened the doors for significant research support. Three PhD students on the project are now designing medical devices for the anesthesia industry. A fourth student is developing software for a hospital corporation."

The survey showed a relatively good match between the stated APSF priority research areas, applications, and grant funding. Survey respondents were asked to classify their applications according to APSF research priority areas. The APSF priority research areas that were most commonly funded are illustrated in Figure 1 and include

- Identification of predictors of patients at increased risk for mishaps (40% of applications, 48% of funded studies)
- New clinical methods for prevention or early diagnosis of mishaps (28% of applications, 21% of funded studies)
- Evaluation of new or re-evaluation of old technologies for prevention and diagnosis of mishaps (28% of applications, 15% of funded studies)
- Development of innovative methods for study of low-frequency events (20% of applications, 30% of funded studies)
- Innovative methods of education and training in safety (11% of applications, 18% of funded studies).

Survey responses showed that applications and funded studies were not restricted to a focus on relatively healthy patients. Survey respondents reported that their applications and funded studies were nearly evenly split between a focus on relatively healthy patients (28% of applications; 30% of funded studies) and special patient populations such as pediatrics or the elderly (29% of applications; 30% of funded studies).

See "Grant Program," Next Page

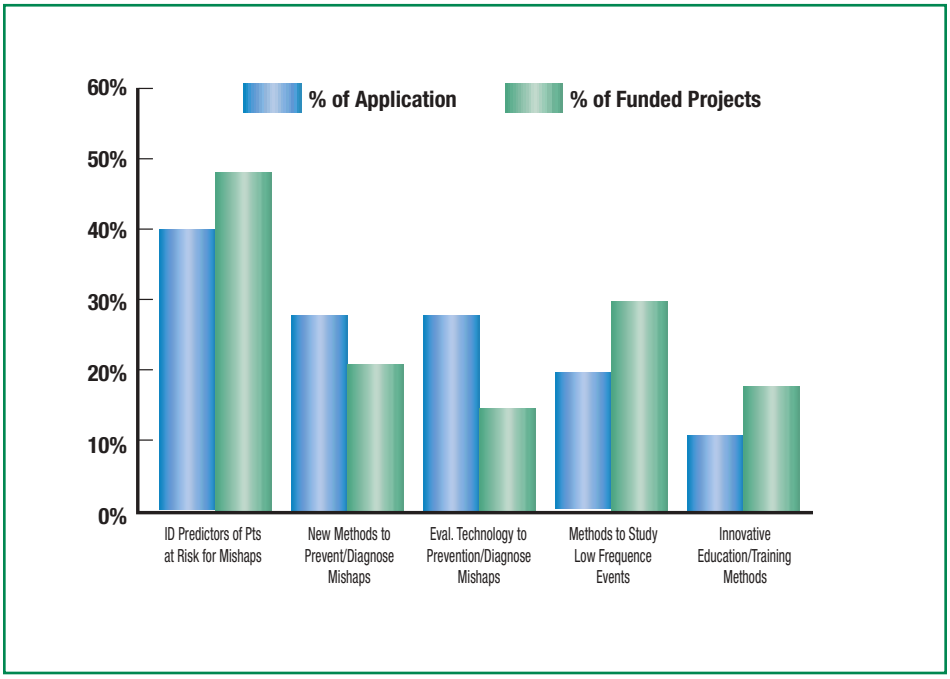

Figure 1. *Most commonly funded research areas.*

48 Projects Result in 214 Publications

"Grant Program," From Preceding Page

The APSF Grant Program lists a number of safety topics appropriate for APSF funding. Applicants classified their projects into multiple categories. The most common topics of projects are shown in Figure 2 and include

- Outcomes or incident measurement (38% of applications, 42% of funded studies)
- Monitoring, effectiveness, or injury prevention (36% of applications, 21% of funded studies)
- Risk assessment or risk factors (29% of applications, 39% of funded studies)
- Human factors or performance (27% of applications, 39% of funded studies)
- Prevention of a specific complication or injury (25% of applications, 30% of funded studies)
- Simulation or computer modeling (15% of applications, 30% of funded studies).

Most funded studies involved clinical trials, non-clinical research involving human subjects, database analysis or simulation, or computer modeling. Few applications or funded studies involved laboratory, bench science, or medical records review.

Improving Patient Safety

Grant recipients reported that the APSF Grant Program had a positive impact on patient safety in anesthesia and beyond. This impact consisted of direct improvements in anesthesia patient safety as well as enhancement of careers in patient safety research that would contribute toward additional patient safety improvements.

APSF grant funding was instrumental in development of successful careers in anesthesia patient safety research for APSF funded investigators as well as other research team members. Grant recipients commented that the APSF was the only funding source for patient safety research in the early years of the program. As one respondent aptly stated, "Without the APSF, I would never have been able to become a successful patient safety researcher since pre-IOM report there were really no other sources of funding for anesthesia patient safety research." Many respondents commented on the importance of APSF funding as "seed money" that led to careers and additional funding for patient safety research. One respondent commented, "APSF funding created an active research team. One member has developed an innovative ICU ventilator. Another directs a simulation center. A third has developed a respiratory monitor for anesthesia. I have continued in anesthesia research for 15 years." Another respondent commented that, "The APSF funding opened the doors for significant research support. Three PhD students on the project are now designing medical devices for the anesthesia industry. A fourth student is developing software for a hospital corporation." Another respondent's comments are representative of many others, "Impossible to have done this work without APSF funding . . . (it) made me a permanent patient safety researcher."

Many projects funded through the APSF Grant Program led to direct improvements in anesthesia patient safety. The APSF Grant Program was instrumental in development of simulators for anesthesia, ACLS, critical care, hemodynamics, sedation, and bioterrorism management. Funding of APSF projects legitimized human factors research in clinical anesthesia, introduced human factors and crisis resource management (CRM) concepts and training in anesthesia and medicine, improved risk analysis methodology and the transfer of these methods to other fields such as trauma care and medical devices, and raised public policy issues regarding sleep and work schedules for residents. APSF funding has influenced device development including anesthesia workstations, ICU ventilators, respiratory monitors, and simulation devices, as well as contributing toward new ways of processing signals and displaying clinical data on monitors. APSF-funded researchers commented on other specific clinical safety contributions resulting from their APSF funded projects. These researchers reported that the APSF Grant Program:

- Was largely responsible for making perioperative normothermia a standard of care
- Raised awareness of postoperative cognitive deficit after non-cardiac surgery and in elderly patients
- Confirmed safety and cost-effectiveness of fast-track cardiac anesthesia
- Reduced concerns about pediatric URI history and risk of airway complications during and after general anesthesia
- Reduced concerns about the need for ambulatory patients to void before discharge
- Influenced ASA practice parameters and perioperative practices
- Raised awareness of the need to review anesthetic emergency protocols.

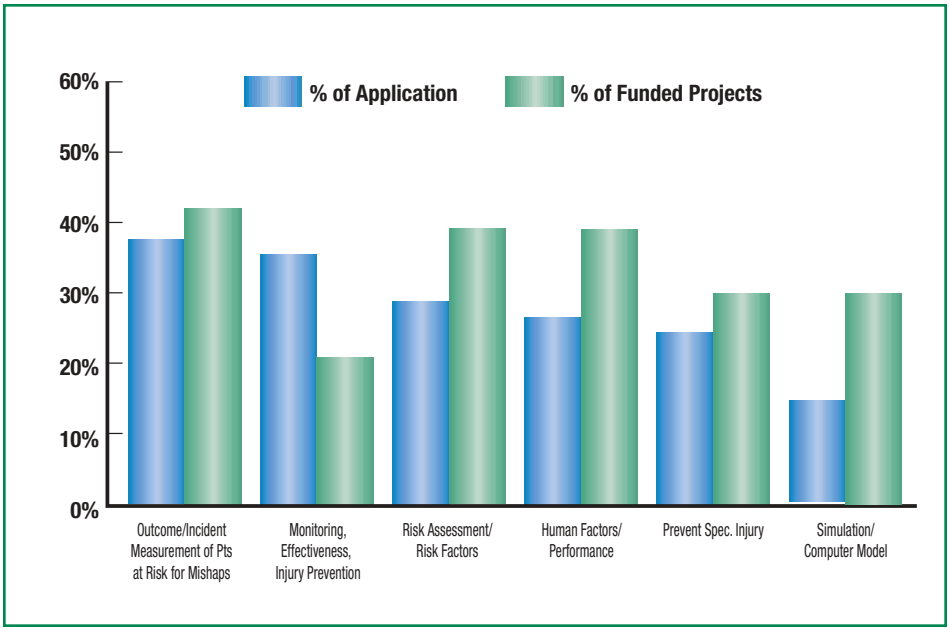

Figure 2. Most common research topics.

Publication and Dissemination of Anesthesia Patient Safety Research Results

Nearly all studies funded through the APSF Grant Program resulted in publication of their results. Only 2 studies funded prior to 1999 did not result in any publications. In addition to publication of APSF-funded project results, APSF funding contributed to additional publications beyond the initial project reporting. The 48 projects funded through the APSF Grant Program have resulted in publication of 214 publications that incorporate or discuss APSF-funded project results.

Criticisms of the APSF Grant Program

It is not surprising that few prior APSF Grant Program applicants who did not received APSF funding for their projects responded to the survey. Among the 84 responses were a number of comments indicating areas for improvement in the program. Most comments addressed feedback on

See "Grant Program," Next Page

Letter to the Editor

Barium ...

Is It Time to Say Goodbye?

To the Editor:

Recent reports of circuit fires following exposure of sevoflurane to desiccated carbon dioxide absorbent has refocused attention on the risks associated with volatile anesthetics. Volatile anesthetics have been in use for more than 150 years, and it is surprising to many clinicians when "new" complications seem to come to light every few years. In the past 20 years, carbon dioxide absorbents have been implicated in a number of these complications. Although we perform daily machine and circuit checks, and most of us have a good working knowledge of how our machines and ventilators work, the CO_2 absorbent rarely gets much attention, unless of course, its color changes, marking exhaustion of its CO_2 absorbing capability.

One of the job descriptions of an anesthesiologist is that of trend watcher, and a trend involving barium-containing CO_2 absorbers has slowly come to light. Exposure of sevoflurane to CO_2 absorbent results in Compound A formation. Desflurane is associated with carbon monoxide formation when exposed to desiccated CO_2 absorbent, and the recent case reports of extreme heat during sevoflurane use have all been associated with the exposure of agent to CO_2 absorbent. All of these risks are increased when barium is the basis of the CO_2 absorbent:

- Barium-based absorber dehydration increases the concentration of Compound A; whereas soda lime dehydration decreases it.[1]
- Desiccated barium-based absorbers are associated with a nearly 7-fold increase in carbon monoxide production (11600 ppm vs. 1800 ppm) versus soda lime when exposed to clinically used concentrations of desflurane.[2]
- All of the 4 reported cases of circuit fires associated with sevoflurane and dessicated CO_2 absorbent involved barium, and a recent laboratory report described how the CO_2 absorber canister exploded and burst into flames when dehydrated barium-based absorber was exposed to sevoflurane.[3]

Barium and soda lime neutralize exhaled CO_2 via an exothermic reaction with a strong base: the former via barium hydroxide, and soda lime via sodium and potassium hydroxide. Both have approximately equal absorbing capabilities, and their cost is similar. Amsorb, a newer agent which contains neither potassium, barium, nor sodium has been proposed as a safer alternative, but it has not yet reached wide market acceptance.

The history of anesthesia is littered with products that fell out of practice for reasons of risk to patient safety. We no longer use ether or cyclopropane due to the risk of explosion. We no longer use hanging bellows due to the risks of decreased circuit leak detection. We no longer use droperidol due to the risk of QT prolongation. All, however, fell out of practice only when better substitutes were available. Soda lime is an excellent CO_2 absorbent and is as efficient as barium-containing absorbers; however, soda lime is less likely to be associated with these rare but real risks when used with the newer volatile anesthetics. It is time to reassess our use of this product, and add it to our list of anesthesia museum pieces.

Roy G. Soto, MD
Tampa, FL

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Improved APSF website:
www.apsf.org

APSF Executive Committee Invites Collaboration

From time to time the Anesthesia Patient Safety Foundation reconfirms its commitment of working with all who devote their energies to making anesthesia as safe as humanly possible. Thus, the Foundation invites collaboration from all who administer anesthesia, and all who provide the settings in which anesthesia is practiced, all individuals and all organizations who, through their work, affect the safety of patients receiving anesthesia. All will find us eager to listen to their suggestions and to work with them toward the common goal of safe anesthesia for all patients.

Your APSF President - Robert K. Stoelting, MD

"Grant Program," From Preceding Page

applications. Other comments addressed clarity (or lack of clarity) regarding funding priorities and disagreement with APSF priorities or focus.

The most common criticisms of the program concerned feedback on unsuccessful applications. Of the 84 applicants not receiving APSF funding who responded to the survey, 9 received either no feedback on their applications or felt the feedback they did receive was not useful. Other respondents felt the APSF should clarify its research interests and perhaps broaden them. Some applicants felt misunderstood, including applicants who received funding for their projects from other sources.

The APSF Scientific Evaluation Committee has recently revised its grant guidelines to include more information on application scoring. An attempt has been made to improve the quality of feedback provided to unsuccessful applicants. These changes may address some of the criticisms expressed by survey respondents.

Conclusions

Over its first 15 years, the APSF Grant Program has stimulated anesthesia patient safety research directly through project funding and indirectly by stimulating careers in anesthesia patient safety research. Projects started with APSF funding have led to continued research in anesthesia patient safety by providing the necessary initial results as well as providing legitimacy to patient safety research. APSF-funded researchers can point out direct impacts on anesthesia patient safety as well as application to patient safety in general.

Dr. Posner is Vice-Chair of the APSF Scientific Evaluation Committee and is a Research Associate Professor in the Department of Anesthesiology at the University of Washington in Seattle.

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The Safe Use of Epidural Steroid Injections

by Stephen E. Abram, MD

Injection of suspensions of corticosteroids into or adjacent to the spinal canal is performed on a regular basis in the United States. Translaminar epidural steroid injections are performed at the lumbar, thoracic, and cervical levels. Caudal injections are done by a "single shot" needle technique as well as by fluoroscopically-guided catheters that are directed toward a particular nerve root. Transforaminal steroid injections (also known as selective nerve root blocks) are performed at nearly every spinal level. Facet joint injections of steroid and local anesthetic are performed routinely in the lumbar and cervical regions. It is not uncommon for injected material to flow into the epidural space, either through rupture of the capsule during injection or via existing capsular tears.

Neuraxial steroid injections are generally considered to be safe by physicians in the U.S. On the other hand, sensationalized reporting of a patient's claim of arachnoiditis resulting from a steroid epidural nearly resulted in abandonment of epidural steroid injections in Australia. As it turned out, the arachnoiditis had been documented on MRI prior to the procedure.

A thorough review of the literature published in 1996 reported a very low incidence of serious complications.[1] Unfortunately, a literature search may not be the best method to determine the true incidence of complications. A somewhat more realistic survey of complications can be found by reviewing the report on the ASA Closed Claims Project published by Fitzgibbon et al., who found that 42% of all claims associated with chronic pain treatment were for complications related to epidural steroid injections (N=114) or facet injections (N=4).[2]

While closed claims analysis helps to identify the types of complications associated with neuraxial steroid injections, it fails to provide a reasonable estimate of the incidence of those complications. We do not know how many and what type of complications occurred with no malpractice claim filed, nor do we know the denominator, i.e., the total number of procedures performed. Another confounding factor is the delay in processing malpractice claims. There is often a delay of months to years in filing a claim, and it may take several years before a claim is litigated or settled. Therefore, we are unaware of most of the complications that have occurred in the past several years, during which time practice may have changed appreciably. For instance, it is likely that a higher proportion of epidural injections are now being done under fluoroscopic guidance, and more patients are treated with a transforaminal approach.

I will briefly review the types of complications that have been reported following neuraxial steroid injections and discuss methods for minimizing risk. I have reviewed published literature, including that obtained from a Medline search subsequent to our 1996 complications review, information from the Closed Claims Analysis, as well as several cases I personally reviewed as an expert witness that have since been settled.

Nerve Injury

Direct Spinal Cord Injury

I have reviewed 2 cases in which spinal cord injury occurred following injection. One case involved a cervical epidural injection performed under fluoroscopy in a deeply sedated patient. The patient suffered a cardiac arrest and was resuscitated, but had severe brain and spinal cord injury and expired following removal of life support. MRI showed injury to the brainstem and cervical spinal cord. The second case involved a lumbar epidural steroid injection done without fluoroscopy. The patient was deeply sedated because she was "allergic" to local anesthetics. Following the procedure she had severe motor and sensory loss in one leg. MRI showed a lesion in the conus.

Hodges et al.[3] reported two cases of nerve injury following cervical epidural steroid injections, both performed in heavily sedated patients using fluoroscopy. In both cases, dural puncture occurred and the needles were repositioned prior to injecting steroids. One patient experienced new persistent painful paresthesias in the upper extremity. The other had new persistent painful paresthesias in one arm and one leg. In both patients, injury to the cord was evident on MRI.

Brouwers et al.[4] reported a spinal cord infarction following a C-6 nerve root injection with iotrolan, bupivacaine, and triamcinolone hexacetonide. The cord injury was documented on MRI scan and resulted in the patient's death. Baker et al.[5] cited this case and 6 other cases of cord injury after transforaminal injections that could not be reported because of pending litigation. They suggested that the mechanism of injury was likely the injection of steroid suspension into a radicular artery with embolization of the spinal cord. They documented visualization of a spinal radicular artery following injection of radiographic dye during a C6-7 transforaminal injection.

Hematoma

I could find only 2 reported cases of neurologic injury associated with hematomas resulting from epidural steroids. One was a report of a subdural hematoma following a cervical epidural steroid injection. The patient had been taking Fiorinal®, which was stopped 2 weeks before the procedure. She developed quadriplegia, recovered partially following surgical decompression, but developed meningitis and died.[6] The other case also occurred after cervical epidural steroid injection. The patient had received 6 previous epidurals over a 2-year period. The patient became quadriplegic, but eventually recovered following extensive decompression surgery.[7] I reviewed the case of a patient who developed upper extremity weakness associated with an epidural hematoma following a cervical epidural steroid injection. Surgical decompression relieved his symptoms, but he developed a recurrent hematoma and experienced permanent upper extremity weakness despite a second operation. He had been on no anticoagulant or antiplatelet drugs. I also reviewed the case of a patient who developed motor and sensory loss after a lumbar epidural steroid injection. He had undergone myelography the day before, which was interpreted as disc herniation. At surgery, he was found to have an epidural angiofibroma and a significant subarachnoid hemorrhage. His recovery was complete. This case occurred prior to the routine use of MRI.

Horlocker et al.[8] prospectively assessed 1035 patients undergoing a total of 1214 epidural steroid injections for the development of neurologic dysfunction associated with hematoma formation. Blocks were performed at the cervical level in 107 procedures, thoracic in 15, lumbar in 988, and caudal in 104. A history of bleeding or bruising was elicited in 176 patients, and 383 patients were taking NSAIDS, with aspirin being the most prevalent. None were taking clopidogrel or ticlopidine. No patients experienced neurological dysfunction requiring assessment for a hematoma.

The Closed Claims Project reported a total of 14 claims of spinal cord injury following epidural steroid injection and 1 following cervical facet injection. The report did not specify the mechanism of spinal cord injury in most cases, although it was stated that 2 cases involved hematomas in patients receiving anticoagulants. An additional 14 patients had non-spinal cord nerve injury, but the exact nature of these was not reported.

Infection

In our 1995 literature review, we found only 2 cases of epidural abscess reported following epidural steroid injection, both in diabetic patients.[1] One patient recovered uneventfully, the other died. Knight et al.[9] subsequently reported an additional 6 cases, most of which were in diabetics. We found reports of 2 cases of meningitis after epidural steroids. In 1 case a dural puncture was documented, and in the other case dural puncture could not be ruled out. The Closed Claims Project reported an additional 12 cases of meningitis and 7 cases of epidural abscess as well as 2 cases of osteomyelitis. No details were reported for any of these cases.

See "Epidural," Next Page

Epidural Suggestions May Reduce Risks

"Epidural," From Preceding Page

Inflammatory Complications

A few case reports of adhesive arachnoiditis were reported following multiple intrathecal injections of methylprednisolone acetate in patients with multiple sclerosis.[1] This led to warnings in the neurology literature about the potential hazards of epidural steroid injections despite the fact that no cases have been reported after *epidural injections*. Indeed, I have found no reports of arachnoiditis after only 1 intrathecal steroid injection. There have been several reports of aseptic meningitis after intrathecal deposteroid injections. Symptoms include fever, nausea and vomiting, lower extremity pain, and, in one report, seizures. CSF examination shows elevated leucocytes and protein and decreased glucose. Cultures are negative. To my knowledge, no cases have gone on to permanent neurologic dysfunction or increased pain. There are no reported cases after epidural steroid injections.

Other Complications and Side Effects

Systemic steroid-induced side effects, including fluid retention, hypertension, congestive heart failure, facial edema, buffalo hump, supraclavicular fat pads, easy bruising, and scaly skin can occur after depo steroid injections. In most cases these changes are dose-related, occurring mainly in patients who receive multiple injections. Cushingoid symptoms can be long-lasting, even when injections are discontinued.[10] Hyperglycemia is commonly seen for several days after the procedure in diabetic patients. Exacerbation of radicular symptoms is common but rarely prolonged. Exacerbation of epidural lipomatosis, severe enough to require surgical decompression, was reported following a series of 3 epidural steroid injections.[11]

Nine cases of death or brain damage were reported in the Closed Claims Project.[2] Five were the result of unintended intrathecal local anesthetic injection, while 3 involved delayed respiratory depression from the addition of morphine to the epidural. A severe allergic reaction accounted for the other case.

Safety Recommendations

While risks are probably small, catastrophic complications can occur after steroid epidurals. Following are suggestions that may reduce risks to patients and may help protect physicians from negligence claims:

1. Provide detailed informed consent. Inform diabetic patients about increased risk of infection as well as the probability of hyperglycemia. Discuss post-procedure diabetes care with the patient and with the primary care physician if pre-procedure management is difficult.
2. Take a careful history; ask about the use of antiplatelet drugs and anticoagulants. Include lay terms such as "blood thinners" and "heart medications." Avoid epidurals in patients on newer antiplatelet drugs such as clopidogrel, ticlopidine, and low molecular weight heparin. Ask about recent bacterial infections. Look for contraindications to corticosteroids.
3. Perform a physical examination. Document preexisting neurologic abnormalities. Look for skin bruising.
4. Do not perform the procedure for improper indications. The procedure is most effective for patients with well-documented radiculopathy. It is generally ineffective for axial low back or neck pain.
5. Consider the use of fluoroscopy. Check for allergy to contrast materials. Inject radiographic dye "live" to rule out intravascular, and particularly, intra-arterial injection. This should definitely be done for transforaminal injections.
6. Rule out intrathecal needle placement with a local anesthetic test dose. Abandon the procedure if dural puncture is evident. Do not attempt the procedure at another level at that time. Allow time for the dural puncture to heal before reattempting.
7. Minimize the amount of steroid used. There is probably no reason to use more than 80 mg methylprednisolone acetate or its equivalent for epidural injection. Lower doses are appropriate for transforaminal injections. Wait at least 2 weeks before considering a repeat injection of steroids at any site. Do not routinely perform a "series" of injections, but tailor therapy to the patient's response. A single injection may be adequate.
8. Limit the total local anesthetic to an amount that is safe if delivered intrathecally. Provide close monitoring initially, which should be continued until total recovery if an intrathecal injection occurs. Use a short-acting local anesthetic, especially in outpatients.
9. Avoid the addition of epidural opioids, especially morphine.

The performance of epidural and transforaminal steroid injections is part of the practice of medicine, and should only be performed by those who are actively (not necessarily exclusively) involved in the practice of pain medicine. Patients who are candidates for these injections deserve careful assessment and attention to technical detail during the procedure.

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Inside:

S AFETY
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Inside:
President Reports on State of the APSF ..... Pg 46
ASA Papers Highlight Safety ..... Pg 49
APSF Awards Three New Grants ..... Pg 50
APSF Grant Program Guidelines ..... Pg 53
Bar Coding May Reduce Drug Errors ..... Pg 54
APSF Workshop Explores HRO Model ..... Pg 55

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